APPENDIX B: PENDING CLAIMS FOLLOWING ENTRY OF THE INSTANT AMENDMENT

1. A method for treating a human subject with a hyperproliferative disease comprising the steps of:

(i) identifying a subject with a hyperproliferative disease characterized by alteration or increased expression of a self gene product in at least some of the hyperproliferative cells in said subject; and

(ii) intradermally administering to said subject an expression construct in a viral particle comprising a self gene under the control of a promoter operable in eukaryotic dendritic cells, wherein the dendritic cells are infected by said construct,

whereby said self gene product is expressed by dendritic cells and presented to immune effector cells, thereby stimulating an anti-self gene product response.

2. The method of claim 1, wherein said self-gene product is an oncogene.

3. The method of claim 2, wherein said oncogene is selected from the group consisting of tumor suppressors, tumor associated genes, growth factors, growth-factor receptors, signal transducers, hormones, cell cycle regulators, nuclear factors, transcription factors and apoptic factors.

4. The method of claim 3, wherein said tumor suppressor is selected from the group consisting of Rb, p53, p16, p19, p21, p73, DCC, APC, NF-1, NF-2, PTEN, FHIT, C-CAM, E-cadherin, MEN-I, MEN-II, ZAC1, VHL, FCC, MCC , PMS1, PMS2, MLH-1, MSH-2, DPC4, BRCA1, BRCA2 and WT-1.

5. The method of claim 3, wherein said growth-factor receptor is selected from the group consisting of FMS, ERBB/HER, ERBB-2/NEU/HER-2, ERBA, TGF-β receptor, PDGF receptor, MET, KIT and TRK.

6. The method of claim 3, wherein said signal transducer is selected from the group consisting of SRC, ABl, RAS, AKT/PKB, RSK-1, RSK-2, RSK-3, RSK-B, PRAD, LCK and ATM.

7. The method of claim 3, wherein said transcription factor or nuclear factor is selected from the group consisting of JUN, FOS, MYC, BRCA1, BRCA2, ERBA, ETS, EVII, MYB, HMGI-C, HMGI/LIM, SKI, VHL, WT1, CEBP-α, NFKB, IKB, GL1 and REL.

8. The method of claim 3, wherein said growth factor is selected from the group consisting of SIS, HST, INT-1/WT1 and INT-2.

9. The method of claim 3, wherein said apoptic factor is selected from the group consisting of Bax, Bak, Bim, Bik, Bid, Bad, Bcl-2, Harakiri and ICE proteases.

10. The method of claim 3, wherein said tumor associated gene is selected from the group consisting of CEA, mucin, MAGE and GAGE.

11. The method of claim 4, wherein said tumor suppressor product is p53.

12. The method of claim 1, wherein said expression construct is a viral vector.

13. The method of claim 12, wherein said viral vector is an adenoviral vector, a retroviral vector, a vaccinia viral vector, an adeno-associated viral vector, a polyoma viral vector, an alphavirus vector, or a herpesviral vector.

14. The method of claim 13, wherein said viral vector is an adenoviral vector.

15. The method of claim 14, wherein said adenoviral vector is replication-defective.

16. The method of claim 15, wherein the replication defect is a deletion in the E1 region of the virus.

17. The method of claim 16, wherein the deletion maps to the E1B region of the virus.

18. The method of claim 17, wherein the deletion encompasses the entire E1B region of the virus.

19. The method of claim 18, wherein the deletion encompasses the entire E1 region of the virus.

20. The method of claim 1, wherein said promoter is selected from the group consisting of CMV IE, human or murine dectin-1, human or murine dectin-2, human CD11c, mammalian F4/80 and human or murine MHC class II.

21. The method of claim 20, wherein said promoter is CMV IE.

22. The method of claim 1, wherein said expression vector further comprises a polyadenylation signal.

23. The method of claim 1, wherein said hyperproliferative disease is cancer.

24. The method of claim 23, wherein said cancer is selected from the group consisting of lung, head, neck, breast, pancreatic, prostate, renal, bone, testicular, cervical, gastrointestinal, lymphoma, brain, colon, skin and bladder.

25. The method of claim 1, wherein said hyperproliferative disease is selected from the group consisting of RA, IBD, OA, leiomyomas, adenomas, lipomas, hemangiomas, fibromas, melanomas, restenosis, pre-neoplastic lesions in the lung and psoriasis.

26. The method of claim 1, wherein said expression construct is administered via injection.

27. The method of claim 26, further comprising multiple injections.

28. The method of claim 26, wherein the injection is performed local to a hyperproliferative or tumor site.

29. The method of claim 26, wherein the injection is performed regional to a hyperproliferative or tumor site.

30. The method of claim 26, wherein the injection is performed distal to a hyperproliferative or tumor site.

31. The method of claim 1, wherein intradermal administration is via continuous infusion.

33. The method of claim 1, wherein said immune effector cells are CTLs.

34. The method of claim 1, further comprising administering to said subject at least a first cytokine.

35. The method of claim 34, further comprising administering to said subject a second cytokine, different from said first cytokine.

36. The method of claim 34, wherein said cytokine is selected from the group consisting of GM-CSF, IL-4, C-KIT, Steel factor, TGF-β, TNF-α and FLT3 ligand.

37. The method of claim 34, wherein said cytokine is administered as a gene encoded by said expression construct.

61. A method for treating a subject with a hyperproliferative disease comprising the steps of:

(i) identifying a subject with a hyperproliferative disease characterized by alteration or increased expression of a self gene product in at least some of the hyperproliferative cells in said patient;

(ii) obtaining a dendritic cell from said patient;

(iii) infecting said dendritic cell *ex vivo* with an expression construct comprising a self gene under the control of a promoter operable in eukaryotic dendritic cells; and

(iv) administering said infected dendritic cell to said subject,

whereby said self gene product is expressed by dendritic cell and presented to an immune effector cell, thereby stimulating an anti-self gene product response.

62. The method of claim 61, wherein said self-gene product is an oncogene.

63. The method of claim 62, wherein said oncogene is selected from the group consisting of tumor suppressors, tumor associated genes, growth factors, growth-factor receptors, signal transducers, hormones, cell cycle regulators, nuclear factors, transcription factors and apoptic factors.

64. The method of claim 63, wherein said tumor suppressor is selected from the group consisting of Rb, p53, p16, p19, p21, p73, DCC, APC, NF-1, NF-2, PTEN, FHIT, C-CAM, E-cadherin, MEN-I, MEN-II, ZAC1, VHL, FCC, MCC , PMS1, PMS2, MLH-1, MSH-2, DPC4, BRCA1, BRCA2 and WT-1.

65. The method of claim 63, wherein said growth-factor receptor is selected from the group consisting of FMS, ERBB/HER, ERBB-2/NEU/HER-2, ERBA, TGF-β receptor, PDGF receptor, MET, KIT and TRK.

66. The method of claim 63, wherein said signal transducer is selected from the group consisting of SRC, ABl, RAS, AKT/PKB, RSK-1, RSK-2, RSK-3, RSK-B, PRAD, LCK and ATM.

67. The method of claim 63, wherein said transcription factor or nuclear factor is selected from the group consisting of JUN, FOS, MYC, BRCA1, BRCA2, ERBA, ETS, EVII, MYB, HMGI-C, HMGI/LIM, SKI, VHL, WT1, CEBP-α, NFKB, IKB, GL1 and REL.

68. The method of claim 63, wherein said growth factor is selected from the group consisting of SIS, HST, INT-1/WT1 and INT-2.

69. The method of claim 63, wherein said apoptic factor is selected from the group consisting of Bax, Bak, Bim, Bik, Bid, Bad, Bcl-2, Harakiri and ICE proteases.

70. The method of claim 63, wherein said tumor associated gene is selected from the group consisting of CEA, mucin, MAGE and GAGE.

71. The method of claim 64, wherein said tumor suppressor product is p53.

72. The method of claim 61, wherein said expression construct is a viral vector.

73. The method of claim 72, wherein said viral vector is an adenoviral vector, a retroviral vector, a vaccinia viral vector, an adeno-associated viral vector, a polyoma viral vector, an alphavirus vector, or a herpesviral vector.

74. The method of claim 73, wherein said viral vector is an adenoviral vector.

75. The method of claim 74, wherein said adenoviral vector is replication-defective.

76. The method of claim 75, wherein the replication defect is a deletion in the E1 region of the virus.

77. The method of claim 76, wherein the deletion maps to the E1B region of the virus.

78. The method of claim 77, wherein the deletion encompasses the entire E1B region of the virus.

79. The method of claim 78, wherein the deletion encompasses the entire E1 region of the virus.

80. The method of claim 61, wherein said promoter is selected from the group consisting of CMV IE, human or murine dectin-1, human or murine dectin-2, human CD11c, mammalian F4/80 and human or murine MHC class II.

81. The method of claim 80, wherein said promoter is CMV IE.

82. The method of claim 61, wherein said expression vector further comprises a polyadenylation signal.

83. The method of claim 61, wherein said hyperproliferative disease is cancer.

84. The method of claim 83, wherein said cancer is selected from the group consisting of lung, head, neck, breast, pancreatic, prostate, renal, bone, testicular, cervical, gastrointestinal, lymphoma, brain, colon, skin and bladder.

85. The method of claim 61, wherein said hyperproliferative disease is selected from the group consisting of RA, IBD, OA, leiomyomas, adenomas, lipomas, hemangiomas, fibromas, melanomas, restenosis, pre-neoplastic lesions in the lung and psoriasis.

86. The method of claim 61, wherein said dendritic cell is administered via injection.

87. The method of claim 86, wherein said injection is performed intradermally..

88. The method of claim 86, wherein the injection is performed local to a hyperproliferative or tumor site.

89. The method of claim 86, wherein the injection is performed regional to a hyperproliferative or tumor site.

90. The method of claim 86, wherein the injection is performed distal to a hyperproliferative or tumor site.

91. The method of claim 86, wherein administration is via continuous infusion.

92. The method of claim 61, wherein said subject is a human.

93. The method of claim 61, wherein said immune effector cell is a CTL.

94. The method of claim 61, wherein step (iv) further comprises administering to said subject at least a first cytokine.

95. The method of claim 94, further comprising administering to said subject a second cytokine, different from said first cytokine.

96. The method of claim 94, wherein said cytokine is selected from the group consisting of GM-CSF, IL-4, C-KIT, Steel factor, TGF-β, TNF-α and FLT3 ligand.

98. The method of claim 94, wherein said cytokine is administered as a gene encoded by said expression construct.

98. A method for treating a subject with a hyperproliferative disease comprising the steps of:

(i) identifying a subject with a hyperproliferative disease characterized by alteration or increased expression of a self gene product in at least some of the hyperproliferative cells in said patient;

(ii) obtaining a cell from said patient;

(iii) culturing said cell in the presence of one or more cytokines or growth factors that induce said cell to differentiate into a dendritic cell;

(iv) infecting said dendritic cell with an expression construct comprising a self gene under the control of a promoter operable in eukaryotic dendritic cells; and

(v) administering said infected dendritic cell to said subject,

whereby said self gene product is expressed by dendritic cell and presented to an immune effector cell, thereby stimulating an anti-self gene product response.

99. The method of claim 98, wherein said obtained cell is a stem cell, a monocyte or an undifferentiated dendritic cell.

100. The method of claim 98, wherein said self-gene product is an oncogene.

101. The method of claim 100, wherein said oncogene is selected from the group consisting of tumor suppressors, tumor associated genes, growth factors, growth-factor receptors, signal transducers, hormones, cell cycle regulators, nuclear factors, transcription factors and apoptic factors.

102. The method of claim 101, wherein said tumor suppressor is selected from the group consisting of Rb, p53, p16, p19, p21, p73, DCC, APC, NF-1, NF-2, PTEN, FHIT, C-CAM, E-cadherin, MEN-I, MEN-II, ZAC1, VHL, FCC, MCC , PMS1, PMS2, MLH-1, MSH-2, DPC4, BRCA1, BRCA2 and WT-1.

103. The method of claim 101, wherein said growth-factor receptor is selected from the group consisting of FMS, ERBB/HER, ERBB-2/NEU/HER-2, ERBA, TGF-β receptor, PDGF receptor, MET, KIT and TRK.

104. The method of claim 101, wherein said signal transducer is selected from the group consisting of SRC, ABl, RAS, AKT/PKB, RSK-1, RSK-2, RSK-3, RSK-B, PRAD, LCK and ATM.

105. The method of claim 101, wherein said transcription factor or nuclear factor is selected from the group consisting of JUN, FOS, MYC, BRCA1, BRCA2, ERBA, ETS, EVII, MYB, HMGI-C, HMGI/LIM, SKI, VHL, WT1, CEBP-α, NFKB, IKB, GL1 and REL.

106. The method of claim 101, wherein said growth factor is selected from the group consisting of SIS, HST, INT-1/WT1 and INT-2.

107. The method of claim 101, wherein said apoptic factor is selected from the group consisting of Bax, Bak, Bim, Bik, Bid, Bad, Bcl-2, Harakiri and ICE proteases.

108. The method of claim 101, wherein said tumor associated gene is selected from the group consisting of CEA, mucin, MAGE and GAGE.

109. The method of claim 102, wherein said tumor suppressor product is p53.

110. The method of claim 98, wherein said expression construct is a viral vector.

111. The method of claim 110, wherein said viral vector is an adenoviral vector, a retroviral vector, a vaccinia viral vector, an adeno-associated viral vector, a polyoma viral vector, an alphavirus vector, or a herpesviral vector.

112. The method of claim 111, wherein said viral vector is an adenoviral vector.

113. The method of claim 112, wherein said adenoviral vector is replication-defective.

114. The method of claim 113, wherein the replication defect is a deletion in the E1 region of the virus.

115. The method of claim 114, wherein the deletion maps to the E1B region of the virus.

116. The method of claim 115, wherein the deletion encompasses the entire E1B region of the virus.

117. The method of claim 116, wherein the deletion encompasses the entire E1 region of the virus.

118. The method of claim 98, wherein said promoter is selected from the group consisting of CMV IE, human or murine dectin-1, human or murine dectin-2, human CD11c, mammalian F4/80 and human or murine MHC class II.

119. The method of claim 118, wherein said promoter is CMV IE.

120. The method of claim 98, wherein said expression vector further comprises a polyadenylation signal.

121. The method of claim 98, wherein said hyperproliferative disease is cancer.

122. The method of claim 121, wherein said cancer is selected from the group consisting of lung, head, neck, breast, pancreatic, prostate, renal, bone, testicular, cervical, gastrointestinal, lymphoma, brain, colon, skin and bladder.

123. The method of claim 98, wherein said hyperproliferative disease is selected from the group consisting of RA, IBD, OA, leiomyomas, adenomas, lipomas, hemangiomas, fibromas, melanomas, restenosis, pre-neoplastic lesions in the lung and psoriasis.

124. The method of claim 98, wherein said dendritic cell is administered via injection.

125. The method of claim 124, wherein said injection is performed intradermally..

126. The method of claim 124, wherein the injection is performed local to a hyperproliferative or tumor site.

127. The method of claim 124, wherein the injection is performed regional to a hyperproliferative or tumor site.

128. The method of claim 124, wherein the injection is performed distal to a hyperproliferative or tumor site.

129. The method of claim 124, wherein administration is via continuous infusion.

130. The method of claim 98, wherein said subject is a human.

131. The method of claim 98, wherein said immune effector cell is a CTL.

132. The method of claim 98, wherein step (iv) further comprises administering to said subject at least a first cytokine.

133. The method of claim 132, further comprising administering to said subject a second cytokine, different from said first cytokine.

134. The method of claim 132, wherein said cytokine is selected from the group consisting of GM-CSF, IL-4, C-KIT, Steel factor, TGF-β, TNF-α and FLT3 ligand.

135. The method of claim 132, wherein said cytokine is administered as a gene encoded by said expression construct.